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Measurement of Postsystolic Shortening to Assess Viability and Predict Recovery of Left Ventricular Function After Acute Myocardial Infarction

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OBJECTIVES We sought to determine whether left ventricular (LV) postsystolic shortening in the region of acute myocardial infarction (MI) predicts functional recovery after primary angioplasty.

BACKGROUND Previous studies in experimental animals have shown that postsystolic shortening during temporary coronary occlusion predicts functional recovery after reperfusion.

METHODS Contrast ventriculography was performed on 35 patients with acute MI before and immediately after angioplasty, and one day, one month, three months and one year later. The centerline method was used to measure regional LV wall motion at end systole from all six ventriculograms as well as motion during isovolumic relaxation ($motion_{iso}$) and postsystolic shortening from end systole until the end of contraction. The ventriculograms of 23 patients with normal anatomy were similarly analyzed.

RESULTS Wall motion at end systole improved significantly from baseline to follow-up in the infarct region. Postsystolic shortening at baseline correlated most closely with the recovery of wall motion at three months in patients with anterior infarction ($r = 0.69$, $n = 25$, $p = 0.0001$) but also with recovery at one month and one year. The correlation was slightly less powerful for $motion_{iso}$. Functional recovery could not be predicted from assessment of $motion_{iso}$ and postsystolic shortening in patients with inferior infarction.

CONCLUSIONS In patients with acute anterior MI, analysis of postsystolic shortening in the infarct region predicts the recovery of systolic LV function after reperfusion. Postsystolic shortening represents active contraction and indicates viable myocardium. (J Am Coll Cardiol 2000;35: 1842–9) © 2000 by the American College of Cardiology

The capability to distinguish viable but dysfunctional myocardium from infarct is clinically useful to determine which patients are most likely to derive a functional benefit from revascularization. Early studies suggested that viability could be assessed by measuring the ability of the left ventricle (LV) to increase contraction in response to stimulants such as epinephrine, nitroglycerin, postextrasystolic potentiation or exercise (1–3). More recent studies have shown that viability can be assessed using perfusion imaging or metabolism studies (4,5). While such studies are able to roughly classify patients into "viable" or "not viable" groups, none of these imaging modalities have been able to predict the magnitude of functional recovery.

Two studies in experimental animals have shown a close correlation between postsystolic shortening during acute coronary occlusion and recovery of LV systolic function measured early and late after reperfusion (6,7). These results suggested that postsystolic shortening is an active process reflecting myocardial viability.

Therefore, this study was performed to determine whether analysis of postsystolic shortening in patients with acute myocardial infarction (MI) is useful for assessing viability and for predicting the recovery of systolic LV function after successful revascularization.

METHODS

Patient population. The study comprised 35 patients with acute MI and 23 patients with normal anatomy from the National Toyohashi Higashi Hospital. The infarct patients were selected from a consecutive series of 100 patients who underwent primary angioplasty for treatment of acute MI, had sustained patency on repeat angiography performed 24 h after

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Abbreviations and Acronyms

LV	= left ventricle
MI	= myocardial infarction
$motion_{iso}$	= motion during isovolumic relaxation
%FS	= percent fractional shortening
PTCA	= percutaneous transluminal coronary angioplasty
SD	= standard deviation

angioplasty, had good quality ventriculograms before, immediately after and 24 h after the angioplasty and consented to undergo three additional cardiac catheterizations one month, three months and one year later. Catheterization and revascularization were performed >6 h after onset of pain only if: 1) the electrocardiogram continued to show R waves in the leads overlying the acute MI, and 2) the patient was still suffering from angina, indicating continuing ischemia. Patients who were hemodynamically unstable or who were >80 years old were excluded. Patients who suffered recurrent angina, reinfarction or reocclusion during the one-year follow-up were also excluded. However, patients with silent restenosis of the infarct artery had repeat angioplasty and were retained in the study. All patients were routinely treated with aspirin, long acting nitroglycerin and calcium antagonists for one year after angioplasty. In addition, patients with residual thrombus in the infarct artery after angioplasty received 120,000 U intracoronary urokinase.

The patients with normal anatomy were selected from the records of patients undergoing routine diagnostic cardiac catheterization if they had normal cardiac and coronary anatomy, normal LV function (ejection fraction >55%), normal LV volume (end diastolic volume index <110 ml/m^2) and no history of sudden death.

All ventriculograms were recorded on 35 mm cine film at 60 frames per s. Ventriculography was biplane, in the 30° right anterior oblique and 60° left anterior oblique projections. The present analysis was confined to the former view, which previous studies have shown to provide the best view for analysis of hypokinesis due to coronary occlusion (8,9).

Ventriculographic analysis. During analysis of the infarct patients' ventriculograms, the operator was blinded to the timing of the study (baseline, post angioplasty, etc.). The earliest normal, non-postpremature systolic beat was selected and traced manually frame by frame from end diastole to end diastole. Each contour was entered into a VAX computer using an x-y digitizing tablet. Left ventricular volume was calculated using the area length method (10). End diastole and end systole were identified as the frames of maximum and minimum chamber volume, respectively.

Wall motion was calculated by a modification of the centerline method (11,12). A centerline is constructed midway between envelopes marking the inner and outer bounds of all contours traced through the cardiac cycle (Fig. 1). This approach helps to guide the direction in which

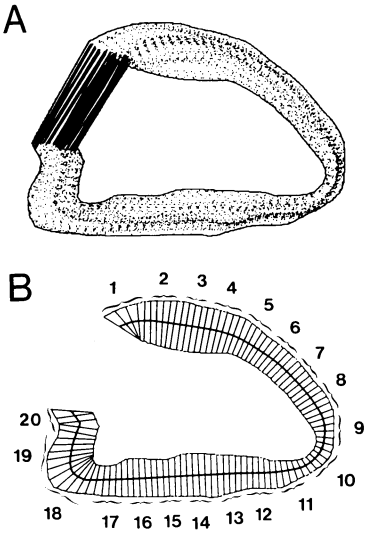

Figure 1. Centerline method for analyzing wall motion through the cardiac cycle. **(A)** Envelopes are constructed demarcating the inner and outer bounds of the LV contours from every angiographic frame in the cardiac cycle. **(B)** A centerline is drawn midway between the inner and outer envelopes. Motion is measured along 100 chords constructed perpendicular to and evenly spaced along the centerline. To simplify the display and analysis, the LV contour is divided into 20 segments by averaging the motion at consecutive sets of five chords. Motion is normalized by the length of the end diastolic perimeter and expressed as a percent. LV = left ventricle.

motion is measured over regions that are akinetic at end systole. One hundred equidistant chords are drawn perpendicular to the centerline and extended to intersect all contours. The magnitude of motion from the first end diastolic border to the border traced from any subsequent frame is measured as distance along these chords. The LV contour was divided into 20 segments whose motion was calculated by averaging the motion of consecutive sets of five chords (13). Motion at each segment was then divided by the end diastolic perimeter length to normalize for patient to patient differences in heart size, and the ratio was expressed as a percent fractional shortening (%FS). The fractional shortening values were converted into units of standard deviation (SD) from the mean of the normal group. Standard deviation units allow function in different regions of the LV to be compared. Using SD unit data, the infarct region was identified as the most abnormally contracting 50% of the territory of the infarct related artery in the baseline, preangioplasty ventriculogram. The severity of hypokinesis due to MI was calculated as the fractional shortening of the five chord segment lying at the center of the infarct region. Functional recovery was calculated as the difference between the baseline fractional shortening of the central infarct segment versus the fractional shortening of the same segment in each of the follow-up studies. The rate of functional recovery was calculated by fitting the measurements of systolic function to the logarithm of time in days.

Motion during isovolumic relaxation ($motion_{iso}$) over the

entire LV contour was measured from the closure of the aortic valve until the opening of the mitral valve and expressed as a %FS. If aortic valve closure was not instantaneous, then the angiographic frame at which the valve first appeared totally closed was selected. Mitral valve opening was the frame in which unopacified blood was first seen to enter the LV chamber. The circumferential extent of shortening during isovolumic relaxation was measured as the percent of the contour having inward motion more abnormal than 2 SD below the normal mean for motion during isovolumic relaxation.

Postsystolic shortening was measured at each individual segment from end systole until that segment reached maximum contraction and expressed as a %FS. In segments whose contraction ended at or before end systole, postsystolic shortening was considered to be zero. Segments whose maximum contraction was smaller than the magnitude of interobserver variability (14) in manually tracing the LV border were also considered to have zero postsystolic shortening. The duration of postsystolic shortening was measured at each segment from end systole until that segment reached maximum contraction. The maximum duration of postsystolic shortening was defined in each patient as the time at which contraction ceased in the last segment to exhibit postsystolic shortening.

Statistical analysis. Linear regression analysis was used to evaluate the ability of $motion_{iso}$ and of postsystolic shortening in the central infarct segment at baseline to predict subsequent recovery of systolic function measured as change in the fractional shortening of that segment. Change in function between studies and between patients with anterior versus inferior MI was evaluated using split-plot design analysis of variance with modification for unbalanced data. Differences between two groups were evaluated using *t* test. Differences between three groups were evaluated using analysis of variance. Values are expressed as the mean ± 1 SD.

RESULTS

Patient populations. The normal patients were mostly men (83%) and had an average age of 57 years (range 45 to 75 years). The duration of isovolumic relaxation was 63 ± 14 ms.

Most of the infarct patients were men (69%), had single vessel disease (60%) and had thrombosed the left anterior descending artery (66%; in nine patients the right coronary artery was affected and in three patients the circumflex). Of those with circumflex thrombosis, two had anterior MIs and one had an inferior MI. The patients also averaged 57 years of age (range 44 to 81 years). The time from onset of symptoms of infarction until revascularization averaged 7.5 ± 7.4 h (range 0.5 to 24). The peak creatine phosphokinase averaged 2,560 ± 2,118 U. The duration of isovolumic relaxation was 98 ± 45 ms ($p < 0.001$ vs. that of normal patients) and was similar in those with anterior and inferior MI (93 ± 30, [n = 25] vs. 110 ± 71 [n = 10] ms, respectively, p = NS).

In the infarct patients, there were four main patterns of wall motion in the infarct region. Some patients had

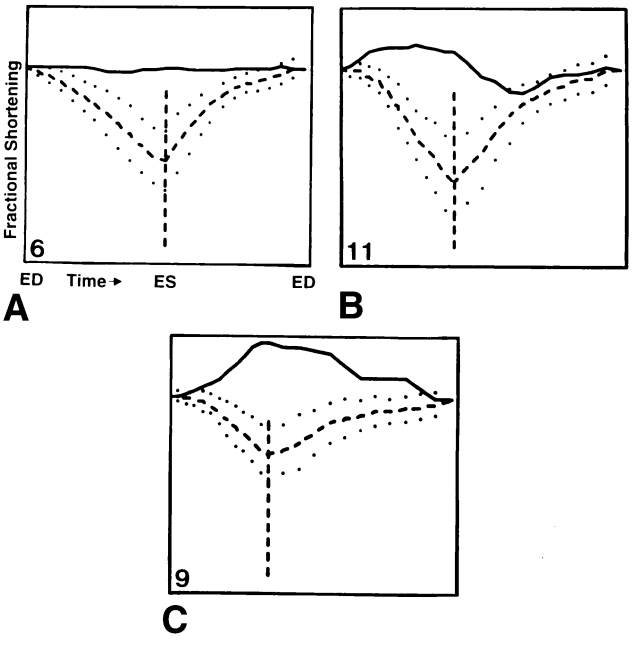

Figure 2. (A) Example of wall motion in an akinetic segment. **(B)** Example of postsystolic shortening with motion of the LV wall to a position inside the end diastolic contour. **(C)** Example of dyskinetic wall motion: inward motion during diastole brings the wall back towards but not within the end diastolic contour. ED = end diastole to end diastole; ES = end systole; LV = left ventricle. **Solid line** = patient's motion; **dashed line** = mean motion of the normal group; **dotted line** = one standard deviation above and below the normal mean. The segment number is displayed.

contraction during systole but subnormal excursion and outward motion during diastole. Some patients had akinesis in the infarct region throughout systole and diastole (Fig. 2A). In others, the LV contour moved inward after end systole to a position inside the end diastolic contour (Fig. 2B). The fourth pattern was dyskinesis throughout systole with just enough inward motion during diastole to return the LV contour to its end diastolic position (Fig. 2C). Since the dyskinesis in the last pattern may be a passive phenomenon, patients whose infarct regions displayed this pattern were examined more closely. All 11 had anterior MI, comprising 44% of this group. Their LV wall motion recovered as much after one year as that of patients without dyskinesis, indicating that the presence of dyskinetic myocardium before revascularization does not rule out viability.

Recovery of systolic function. Wall motion at end systole in the central infarct segment was severely depressed at baseline, particularly in patients with anterior MI. Motion improved significantly ($p < 0.001$, n = all 35 patients) over time after angioplasty in the infarct region, with maximum improvement achieved after one year (Fig. 3 and 4). The severity of dysfunction was worse for anterior MI ($p < 0.002$). The magnitude of functional recovery from baseline did not differ significantly by infarct location although

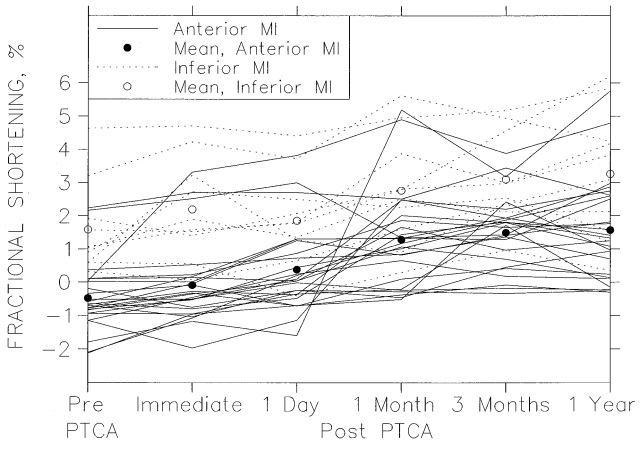

Figure 3. Change in wall motion in the infarct region over time from baseline until one year follow-up study in patients with anterior and inferior myocardial infarction (MI). PTCA = percutaneous transluminal coronary angioplasty.

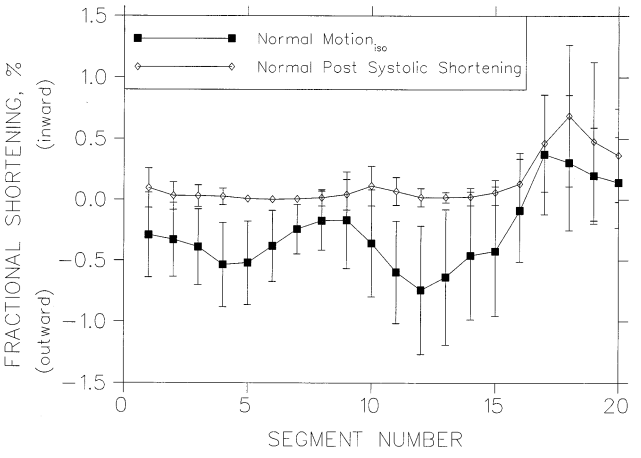

Figure 5. Normal mean and standard deviation for wall motion during isovolumic relaxation and for postsystolic shortening. Motion$_{iso}$ = motion during isovolumic relaxation.

recovery tended to be greater in anterior MI. For example, recovery from baseline to one month after angioplasty (PTCA) was 1.8 ± 1.7 %FS in anterior MI and 1.2 ± 0.9 %FS in inferior MI.

Relationship between baseline function and subsequent recovery. The severity of hypokinesis in the infarct region immediately after angioplasty and one day later was closely related to baseline hypokinesis ($r = 0.87$ for postangioplasty; $r = 0.82$ for day 1, $p < 0.001$ for both). As function recovered subsequently, the correlation decreased ($r = 0.52$, $r = 0.57$, $r = 0.47$, respectively, at one month, three months and one year, $p < 0.005$ for all).

Motion during the isovolumic relaxation period. In the normal patients, average motion$_{iso}$ was outward in all walls except the mitral valve plane (Fig. 5). In infarct patients, motion$_{iso}$ was inward in the infarct region. The principal factor determining the magnitude and circumferential extent of motion$_{iso}$ was infarct location. Motion$_{iso}$ in patients with anterior MI was more depressed (0.5 ± 1.0 %FS [n = 25] vs. 0.16 ± 1.4 %FS [n = 10], respectively, $p < 0.0001$) and more extensive (21 ± 14 vs. 4 ± 7% of the LV contour, respectively, $p < 0.001$) than in inferior MI (Fig. 6). Motion$_{iso}$ was not related to heart rate, LV volume, infarct size as measured from creatine kinase release or time from onset of chest pain until reperfusion.

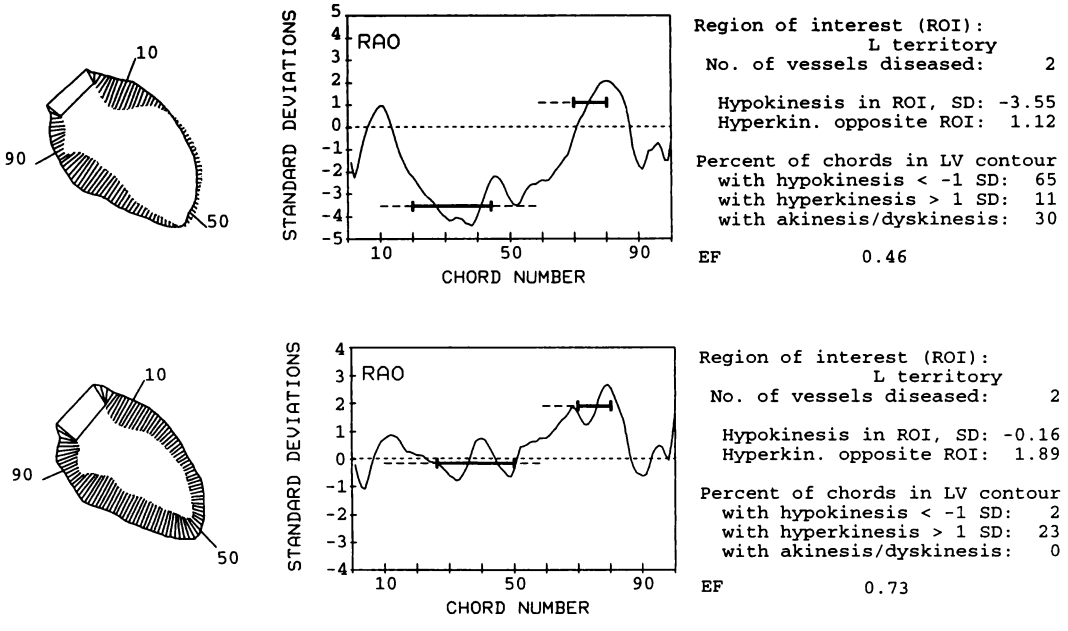

Figure 4. Example of recovery of LV wall motion in a patient with anterior MI. **Top,** Preangioplasty. **Bottom,** Three months after angioplasty. EF = ejection fraction; L = left anterior descending coronary artery; LV = left ventricle; RAO = right anterior oblique; ROI = region of interest; SD = standard deviation.

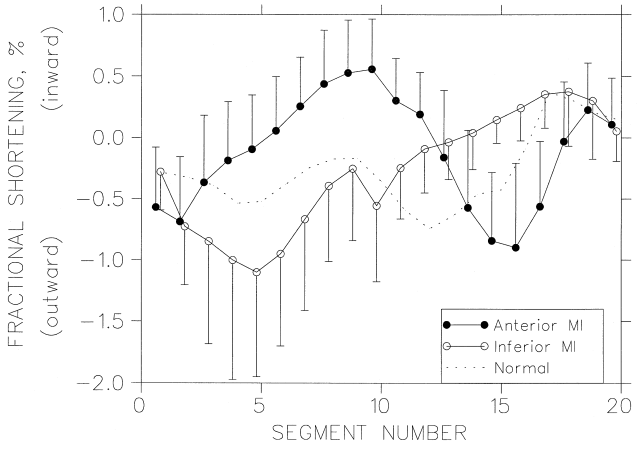

Figure 6. Wall motion in anterior and inferior myocardial infarction (MI) during isovolumic relaxation, compared with normal patients.

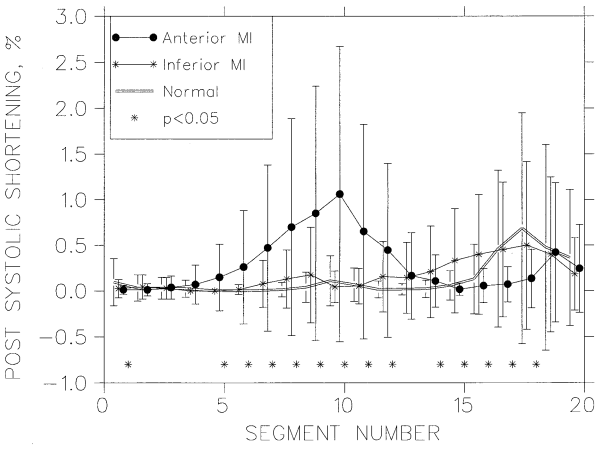

Figure 7. Postsystolic shortening in patients with anterior and inferior myocardial infarction (MI) compared with normal patients. *Left ventricular segments at which shortening values differ significantly ($p < 0.05$) between the three groups by analysis of variance.

Prediction of functional recovery by motion during isovolumic relaxation. Recovery of systolic function, global or regional, at one month, three months and one year after revascularization correlated significantly with the magnitude of $motion_{iso}$ measured from the baseline, pre-PTCA ventriculogram (Table 1). Patients who had more diastolic contraction had a greater recovery. The rate of functional recovery also correlated with $motion_{iso}$ at baseline although less closely ($r = 0.43$, $p < 0.02$ for all patients; $r = 0.45$, $p < 0.05$ for anterior MI). There was no correlation between the circumferential extent of shortening during isovolumic relaxation and functional recovery. Functional improvement immediately after PTCA and one day later was not predicted by $motion_{iso}$.

Postsystolic shortening. In normal patients, there was very little inward motion in diastole, except in the region of the mitral valve (Fig. 5). In the infarct patients there were wide variations in the magnitude of postsystolic shortening (Fig. 7) as evidenced by the standard deviation. Postsystolic shortening peaked at segment 10 ± 2 in anterior MI and at segment 17 ± 2 in inferior MI ($p < 0.001$ vs. anterior); in normal patients it peaked at segment 16 ± 6. The magnitude of postsystolic shortening in the segment with the greatest diastolic contraction averaged 1.3 ± 0.5 %FS in anterior MI and 0.7 ± 0.4 %FS in inferior MI ($p < 0.001$); the measurement in normals was 1.0 ± 0.6 %FS. Since the location of peak postsystolic shortening in anterior MI occurs in a region of the LV that has virtually no postsystolic shortening normally, the significance of the postsystolic shortening observed in anterior MI patients greatly exceeds its numerical value. In inferior MI, both the location and

Table 1. Relationship Between Motion During Isovolumic Relaxation Measured at Baseline and Functional Recovery in the Infarct Region

Time of Follow-up	Patient Population	Number	Correlation Coefficient	p Value
Immediately Post PTCA	All	35	0.04	NS
	Anterior MI	25	−0.09	NS
	Inferior MI	10	0.26	NS
1 day	All	34	−0.34	NS
	Anterior MI	25	−0.24	NS
	Inferior MI	9	−0.17	NS
1 month	All	35	−0.42	0.0110
	Anterior MI	25	−0.44	0.0278
	Inferior MI	10	0.04	NS
3 months	All	35	−0.58	0.0002
	Anterior MI	25	−0.61	0.0012
	Inferior MI	10	−0.30	NS
1 year	All	35	−0.49	0.0031
	Anterior MI	25	−0.57	0.0030
	Inferior MI	10	−0.08	NS

MI = myocardial infarction; NS = not significant ($p > 0.05$); PTCA = percutaneous transluminal coronary angioplasty.

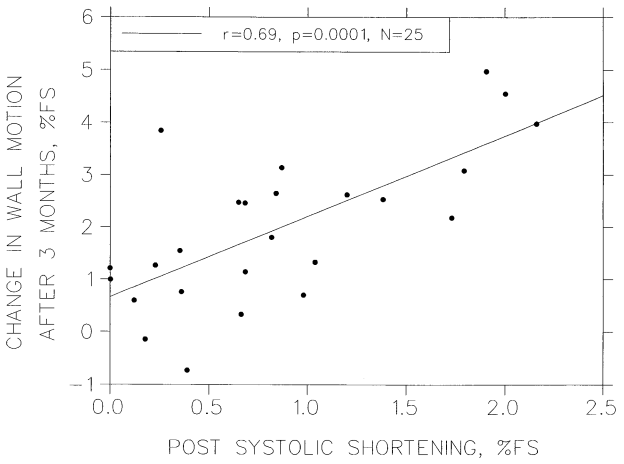

Figure 8. Relationship between recovery of wall motion in the infarct region after three months in patients with anterior myocardial infarction (MI) and peak total postsystolic shortening measured at the baseline, preangioplasty angiogram. %FS = fractional shortening. **Black circles** = data of one patient.

magnitude of peak postsystolic shortening largely overlapped with those of the normal group. The magnitude of postsystolic shortening was not related to heart rate, infarct size as estimated from peak creatine phosphokinase, LV volume or time from onset of chest pain until reperfusion. The duration of postsystolic shortening varied from segment to segment in individual patients. The maximum duration of postsystolic shortening was similar in anterior and inferior MI (121 ± 51 vs. 123 ± 37 ms, respectively, p = NS) and was significantly longer than the duration of isovolumic relaxation (122 ± 47 vs. 98 ± 45 ms, respectively, n = 35 MI patients, $p < 0.02$).

Prediction of recovery by postsystolic shortening. Improvement in wall motion in the infarct region between the baseline ventriculogram and follow-up correlated significantly with postsystolic shortening in patients with anterior MI ($r = 0.69$, $p = 0.0001$ for recovery at three months) (Fig. 8) (Table 2). Patients who displayed more postsystolic shortening had greater recovery. The relationship was stronger than with $motion_{iso}$. In patients with inferior MI, there was no significant correlation between recovery and baseline postsystolic shortening ($r \leq 0.4$ for all). The rate of functional recovery also correlated significantly, although weakly, with postsystolic shortening ($r = 0.45$, $p < 0.01$ in all patients; $r = 0.49$, $p < 0.02$ in anterior MI). Immediate improvement in function after angioplasty and one day later was not predicted by postsystolic shortening.

Improvement in ejection fraction also correlated significantly with postsystolic shortening ($r = 0.56$, $r = 0.44$, $r = 0.42$, respectively, at one month, three months and one year after MI, $p = 0.0005$, $p = 0.0082$, $p = 0.012$).

DISCUSSION

Previous studies of postsystolic shortening. In 1987, Brown et al. (6) reported that experimental animals subjected to coronary artery occlusion display postsystolic shortening during isovolumic relaxation and that the magnitude of postsystolic shortening predicts recovery of systolic function immediately after reperfusion (6). In 1988, this investigative team extended their findings to predictions of functional recovery at two to three weeks after reperfusion (7). The present study is the first to attempt to substantiate this finding in patients. The results show that the magnitude of postsystolic shortening measured before reperfusion correlates significantly with late recovery of regional wall motion within the infarct region.

Table 2. Relationship Between Postsystolic Shortening at Baseline and Functional Recovery in the Infarct Region

Time of Follow-up	Patient Population	Number	Correlation Coefficient	p Value
Immediately Post PTCA	All	35	0.03	NS
	Anterior MI	25	0.12	NS
	Inferior MI	10	0.03	NS
1 day	All	34	0.37	0.0295
	Anterior MI	25	0.26	NS
	Inferior MI	9	0.38	NS
1 month	All	35	0.49	0.0030
	Anterior MI	25	0.51	0.0100
	Inferior MI	10	−0.05	NS
3 months	All	35	0.66	0.0000
	Anterior MI	25	0.69	0.0001
	Inferior	10	0.35	NS
1 year	All	35	0.57	0.0004
	Anterior MI	25	0.65	0.0005
	Inferior MI	10	0.21	NS

MI = myocardial infarction; NS = not significant ($p > 0.05$); PTCA = percutaneous transluminal coronary angioplasty.

These results support and extend previous studies that have shown that postsystolic shortening is an active contractile process indicative of viable myocardium. Gibson et al. (15) reported that delayed inward motion during isovolumic relaxation, the most common manifestation of asynchrony in patients with acute MI, resolved after restoration of coronary perfusion. Furthermore, the presence of postsystolic shortening was associated with a higher probability of successful reperfusion, suggesting that postsystolic shortening is a sign not only of viable myocardium but also of a viable microcirculation.

Duration of postsystolic shortening. In this analysis, postsystolic shortening from end systole until the end of contraction was measured as well as shortening during isovolumic relaxation. The results showed that, even though the duration of isovolumic relaxation is prolonged in acute MI, postsystolic shortening lasts even longer. Other investigators have also reported that contraction may be prolonged to >200 ms after end systole or to one-third diastole (16,17). In normal patients, the duration of isovolumic relaxation is 116 ± 41.5 ms if defined angiographically as the period from minimum volume of the smoothed volume time curve until the first appearance of unopacified blood in the LV (18). If defined from the closure of the aortic valve to the opening of the mitral valve, the average duration of isovolumic relaxation measured by a number of echocardiographic techniques varies from 39 to 91 ms (mean 62 ± 14) in normal populations (19), similar to that measured in this study. In patients with acute MI, isovolumic relaxation was significantly prolonged. This is attributable to asynchronous contraction, which has been shown to correlate directly with delayed and asynchronous relaxation (16). In experimental animals subjected to acute coronary occlusion, opening of the mitral valve occurred 50 ms after peak negative dP/dt, whereas peak postsystolic shortening was delayed until 75 ms after peak negative dP/dt in the central infarct region (6). Thus, both experimental and clinical studies are in agreement that postsystolic shortening due to acute ischemia is delayed beyond the isovolumic relaxation period.

Factors affecting prediction of functional recovery. In this study, diastolic contraction only predicted functional recovery for patients with anterior MI. That there are regional differences in both the magnitude of postsystolic shortening and its ability to predict recovery has been well documented. In two studies of experimental anterior MI, postsystolic shortening predicted subsequent functional recovery. In one experimental study of posterior MI due to circumflex artery ligation, postejection wall thickening in the posterior wall did not predict recovery (20). Since the latter authors did not study anterior MI, the negative result cannot definitely be attributed to the infarct location. The differential responsiveness of the anterior versus the posterior or inferior walls has been demonstrated under other conditions besides coronary occlusion. For example, two investigative groups found that the combination of calcium antagonist therapy and halothane anesthesia causes a uniform, global depression of systolic shortening, but only the anterior wall develops significant postsystolic shortening (21,22). The authors speculated that the regional response may reflect the underlying myocardial fiber architecture, which is thinner at the apex, or the heterogeneity of normal systolic shortening, which is lowest at the anteroapical region. Hammermeister et al. (18) felt that the delayed onset of contraction normally seen in the anteroapical region explains why its function differs from that in other regions of the LV. They argued that the delayed electrical activation of the apex and its thinner wall make it more difficult for this region to shorten against the afterload generated by the earlier contracting anterobasal and inferior walls. Furthermore, its smaller radius of curvature predisposes the apical region to higher stress, for example, making it susceptible to aneurysm formation. These factors may predispose the anteroapical region to shorten later when LV pressure is falling and the other walls are beginning to move outward.

The fact that postsystolic shortening could not predict immediate functional recovery after reperfusion or one day later can also be attributed to the different conditions of experimental ischemia versus clinical MI. In Brown et al.'s study (6), the duration of coronary occlusion before reperfusion was only 60 or 90 min, whereas in this study patients were revascularized up to 24 h after the onset of pain if they had evidence for continuing ischemia and up to 6 h if they did not. That is, the experimental protocol produced much less severe ischemia from which the myocardium was able to recover active shortening in most cases after only 3 h of reperfusion. In contrast, regional wall motion in our patients' MIs did not recover as rapidly. Although the severity of dyskinesis was diminished by 24 h after angioplasty, active shortening did not return until some time before the one month angiogram. Indeed, the ultra early functional "recovery" observed in this study and in previous clinical studies most probably reflects retraction of dyskinesis (23). Retraction of dyskinesis may be related to other processes, such as stiffening of the infarct region by edema, rather than actual recovery of myocardial contraction, since it is seen in both reperfused and nonreperfused patients.

Differences in hemodynamic status between this study and earlier experimental studies may also have been a factor. The magnitude of postsystolic shortening in ischemic myocardium correlates negatively with coronary perfusion pressure and with preload (24). The patients in this study were conscious. However, the experimental animals in the previous studies were anesthetized with halothane and nitrous oxide, agents that tend to decrease cardiac output and systemic arterial pressure.

Although the correlation observed in this study was highly significant, the r^2 value of 0.48 indicates that viability as indicated by baseline postsystolic shortening explains only half of the functional recovery. One reason for this is that the magnitude of functional recovery is influenced by a number of factors besides viability. Previous studies have shown that the magnitude of recovery is related to the severity of dysfunction present acutely, being greatest in those with the severest defect at baseline (25,26). In this study, the patients all had severe hypokinesis at baseline, were treated early or while ischemia

was still active, had successful primary PTCA and were revascularized for restenosis. Under this rigorous therapeutic regimen, nearly all of them had excellent recovery of wall motion in the infarct region. The lack of treatment failures may have obscured the potential predictive power of measuring postsystolic shortening by limiting the range of values. Another possible factor affecting their recovery is variability in the clinical course of the patients. For example, restenosis of sufficiently long duration and severity in some patients could have induced myocardial hibernation and worsened their ventricular function. A third factor affecting functional recovery is reperfusion injury. Despite these considerations, the results of this study help to confirm previous studies showing that postsystolic shortening is an active process, one that is indicative of myocardial viability.

In summary, postsystolic shortening in patients with acute anterior MI correlates with subsequent recovery of systolic function after revascularization. Postsystolic shortening represents active contraction and indicates viable myocardium.

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